MEDICAL PROCEEDINGS

MEDIESE BYDRAES

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REDAKSIONEEL · EDITORIAL

INENTING TEEN POLIOMIËLITIS

Dit is 'n ope vraag of die uitroeiing van poliomiëlitis, as openbare gesondheidsmaatreël, voorrang behoort te hê bo die maatreëls teen ander gedugte siektes in die gemeenskap. In sy *Jaarverslag* vir 1957 (die jongste verslag wat beskikbaar is) sê die Sekretaris van Gesondheid, byvoorbeeld, dat die witseerkeelposisie gedurende die afgelope 10 jaar feitlik onveranderd gebly het. Die gevolge van witseerkeel is onbenullig in vergelyking met die verwoesting by diegene wat polio oorleef. Nietemin is dit interessant om poliomiëlitissterfgevalle met witseerkeelsterfgevalle te vergelyk. Selfs gedurende tye van epidemiese poliomiëlitis is die verskil opvallend en behoort dit heelwat besorgdheid te baar.

Tydens die poliomiëlitis-epidemie in 1957 was daar 183 sterfgevalle ten gevolge van hierdie siekte, in vergelyking met 277 witseerkeelsterfgevalle.

In 1956 (ook 'n epidemiese polio-jaar) was daar 240 poliosterfgevalle in vergelyking met 'n ‚normale' 324 witseerkeelsterfgevalle.

In 'n nie-epidemiese jaar (1954) was daar net 53 poliosterfgevalle, maar daar was 316 witseerkeelsterfgevalle, d.w.s. daar was 6 keer meer sterfgevalle ten gevolge van witseerkeel as ten gevolge van poliomiëlitis.

Ten spyte van hierdie ontstellende toestand is die verbeelding van die publiek aangegryp deur antipoliomiëlitispropaganda, liewer as deur die voor die hand liggende behoefte om dringende en doeltreffende stappe teen 'n volkome voorkombare siekte soos witseerkeel te doen.

Wat ook al die verdienste van die saak, bly dit 'n feit dat ons vandag onontkombaar be-

VACCINATION AGAINST POLIOMYELITIS

It is a moot point whether, as a public health measure, the eradication of poliomyelitis should enjoy priority over measures against other formidable diseases in the community. In his *Annual Report* for 1957 (the latest available), for example, the Secretary for Health stated that the diphtheria position had remained virtually unchanged over the previous 10 years. The sequelae of diphtheria are inconsiderable in comparison with the ravages in the survivors of paralytic poliomyelitis. Nevertheless it is enlightening to contrast poliomyelitis and diphtheria deaths. Even in times of epidemic poliomyelitis the difference is striking and should give cause for considerable concern.

In the epidemic polio year of 1957 there were 183 deaths from poliomyelitis as against 277 diphtheria deaths.

In 1956 (also an epidemic polio year) there were 240 polio deaths as against the ' normal ' 324 diphtheria deaths.

In a non-epidemic year (1954), there were only 53 polio deaths as against 316 diphtheria deaths, i.e. there were 6 times as many deaths from diphtheria as from poliomyelitis.

Despite this alarming situation, the popular fancy has been caught by antipoliomyelitis propaganda rather than the obvious case to take urgent and effective steps against such a wholly preventable disease as diphtheria.

Whatever the merits of the matter, the fact remains that we are today inescapably involved in a massive national attempt to wipe out poliomyelitis, a campaign dependent on the availability of suitable vaccines. That a safe

trokke is by 'n massiewe uniale poging om poliomiëlitis uit te roei—'n veldtog wat afhanklik is van die beskikbaarheid van geskikte entstowwe. Dat 'n veilige mondelinge entstof met verswakte poliovirusse van die tipes 1, 2 en 3 geproduseer kan word, skyn 'n uitgemaakte saak te wees. Daar is aansienlike produksiemoeilikhede en, heeltemal ten regte, word daar aangedring op voldoening aan strenge maatstawwe voordat hierdie entstof as veilig vir gebruik beskou word.

Terwyl die beskikbaarheid van 'n mondelinge lewendige entstof dit redelik gemaak het om te begin dink aan die moontlikheid om die poliovirus as 'n patogeen uit die maagdermkanaal van die mens te verban, kan daar nie liggies afgesien word van die gebruik van parenterale geïnaktiveerde entstof van die Salk-tipe nie, veral in die huidige oorgangsfase.

As ons doel die algehele eliminasie van poliovirus in die gemeenskap is (of die algehele eliminasie van sy gevare), is die eerste stap massa-vaksinasie so nie van die hele bevolking nie dan wel van dié ouderdomsgroepe wat vir infeksie vatbaar is, deur die toediening van verswakte virusse binne 'n kort tydjie.

„Volgende op hierdie „algehele bedekking" met vaksienvirusse, sal spesiale ondersoek ingestel moet word om die mees uitvoerbare manier vir die handhawing van die bevolking se weerstandskragtigheid vas te stel. Verdere ondersoek sal gedoen moet word om verskille in die biologiese gedrag van die poliovirus onder uiteenlopende klimaatstoestande, in verskillende sosiaalekonomiese groepe, en in gemeenskappe met uiteenlopende geleenthede vir die toediening van virus, soos inrigtings, plattelandse streke, klein dorpe en groot stede, vas te stel."[1]

Die beskikbaarheid van die mondelinge vaksien is op sigself nie voldoende om welslae te verseker nie. Dit is van ewe groot belang om die immunisasieveldtog op die regte tydstip van die jaar van stapel te stuur, en om seker te maak dat daar geen immunologiese leemtes in die bevolking is nie. Die beste resultate is nog altyd behaal wanneer immunisasie tydens die tussen-epidemietydperke aangepak is.[2]

Die eerste fase van 'n immunisasieveldtog is teen die einde van verlede jaar in Suid-Afrika onderneem met 'n eenwaardige tipe-1-soort. Net 'n sekere mate van welslae is met hierdie veldtog behaal. Ongelukkig is dit van stapel gestuur op 'n tydstip van die jaar toe ander virusse vry algemeen was. Die aanwesigheid van hierdie ander virusse in die spysverteringskanaal kan die poliovirus nogal in 'n aansienlike mate verhinder om behoorlik te

1. Komitee van Deskundiges insake Poliomiëlitis, Wêreldgesondheidsorganisasie (1960): *Derde Verslag*, Geneve.
2. WHO Chronicle (1960): **14**, 466.

oral vaccine can be produced with attenuated strains of types 1, 2 and 3 polioviruses seems fairly well established. There are considerable production difficulties and, quite rightly, rigorous standards which must be complied with before a batch can be considered safe for release.

While the availability of an oral live vaccine has made it reasonable to think of possibly eliminating poliovirus from the human gastro-intestinal tract as a pathogen, the use of parenteral (Salk-type) inactivated vaccine is not lightly to be abandoned, especially in the contemporary transition phase.

If our goal is the total elimination of poliovirus in the community (or total elimination of its hazards) the first step is the mass vaccination, if not of the whole population, at least of those age groups most susceptible to infection, by administering attenuated viruses within a short time.

'Following this "blanketing" with vaccine viruses, special studies will have to be conducted to determine the most feasible means of maintaining the resistance of the population. More studies will be needed to determine the differences in biological behaviour of poliovirus under various climatic conditions, in various socio-economic groups, and in communities with differing opportunities for administration of virus, such as institutions, rural areas, small towns and large cities.'[1]

The availability of the oral vaccine is not enough to ensure success. It is equally important to conduct the immunization campaign at the right season of the year, making certain that no immunological gaps are created in the population. The best results have been obtained when immunization was performed in the inter-epidemic period.[2]

The first phase of an immunization campaign was undertaken in South Africa towards the end of last year with a monovalent type 1 strain. This campaign met with a qualified success. Moreover, it was unfortunately begun at a time of the year when other viruses were prevalent. These other viruses in the alimentary tract can interfere substantially with the success of the 'take' of the poliovirus. It is important for the public as well as the profession to be informed of what incidence of success attended the first stage of the campaign. This information should be released without delay.

There is evidence, from the first mass live poliovirus vaccination ever undertaken (in the

1. WHO Expert Committee on Poliomyelitis (1960): *Third Report*. Geneva.
2. WHO Chronicle (1960): **14**, 466.

‚vat.' Dit is van die allergrootste belang dat die publiek sowel as die professie ingelig moet word oor die mate van welslae waarmee die eerste stadium van die veldtog bekroon is. Hierdie inligting moet sonder versuim beskikbaar gestel word.

Die eerste massa-vaksinasie met lewendige poliovirus wat nog ooit onderneem is (in die Belgiese Kongo in 1957)[3] het bewys dat die antiliggaam-reaksie op vaksinasie slegs 60% was, vermoedelik ten gevolge van virusinmenging.

In 1959 in Mexiko[4] het virologiese studies vóór inenting 'n hoë voorkoms van natuurlike enterie-virusinfeksies aangetoon wat 'n hoogtepunt van 72% gedurende die eerste lewensjaar bereik het. Die serologiese omsettingsyfer 10 weke ná 'n enkele dosis van die driewaardige vaksien was 68% vir tipe 1, 82% vir tipe 2, en slegs 43% vir tipe 3. 'n Tweede toediening aan 44 kinders onder 4 jaar wat ná die eerste dosis nie op een of meer tipes gereageer het nie, het 6 weke later omsettingsyfers van 96% vir tipe 1, 96% vir tipe 2 en 72% vir tipe 3 tot gevolg gehad.

Inmenging is derhalwe nie 'n onoorkoomlike struikelblok nie, maar dit vestig die aandag op die noodsaaklikheid om die veldtog gedurende die optimale seisoen van die jaar van stapel te stuur as maksimum-welslae binne minimum-tyd verlang word. So nie sal 'n onbekende persentasie van die gemeenskap 'n valse gevoel van veiligheid hê, met moontlike rampspoedige gevolge vir die onbeskermde persone wat hulle pligsgetrou aan al die immunogeniese maatreëls onderwerp het.

Daar word gehoop dat die versameling van wetenskaplike gegewens oor die krag en doeltreffendheid van die Suid-Afrikaanse entstof nie veronagsaam sal word nie, sodat sowel die professie as die publiek ten volle oor die immunologiese status van die gemeenskap ingelig kan word.

Volgens 'n aanbeveling van die Komitee van Deskundiges van die Wêreldgesondheidsorganisasie moet ten minste 25 drievoudig negatiewes, in die geval van 'n eenwaardige vaksien, ondersoek word vóór en 1–2 maande ná inenting. Indien 'n driewaardige vaksien gebruik word, behoort ongeveer 50 sodanige persone ondersoek te word—en selfs meer, as dit 'n bekende feit is dat ander belemmerende virusse onder die gemeenskap aanwesig is. Die monster moet volgens gesonde statistiese beginsels gekies word.

Verlede jaar se veldtog is gekenmerk deur die sterk klem wat op die skoolgaande bevolking, d.w.s. op kinders 6 jaar en ouer, gelê is. Spesiale voorsiening vir hul immunisasie is by die skole gemaak.

3. Komitee van Deskundiges insake Poliomiëlitis, Wêreldgesondheidsorganisasie (1960): *Derde Verslag*, bl. 11, Geneve.

4. *Ibid.*, bladsy 15.

Belgian Congo in 1957)[3] that the antibody response to vaccination was only 60%, presumably because of viral interference.

In Mexico[4] in 1959, virological studies before vaccination revealed a high incidence of natural enteric viral infections, reaching a peak of 72% during the first year of life. The serological conversion rates 10 weeks after a single dose of the trivalent vaccine were 68% for type 1, 82% for type 2 and only 43% for type 3. A second administration to 44 children under 4 years who had failed to respond to one or more types after the first dose, produced conversion rates 6 weeks later of 96% for type 1, 96% for type 2 and 72% for type 3.

Interference is therefore not an insuperable obstacle, but it points to the need for timing the campaign at an optimal season of the year, to obtain maximum success within a minimum time, otherwise an unknown percentage of the community will have a false sense of security with possibly disastrous results for the unprotected persons who have conscientiously gone through all the immunogenic motions.

It is to be hoped that the gathering of scientific data about the potency and efficacy of the South African vaccine will not be neglected, so that both the profession and the public may be fully aware of the immunological status attained by the community.

In terms of the recommendations of the WHO Expert Committee, in the case of a monovalent vaccine, at least 25 triple negatives should be examined before and 1–2 months after vaccination. If a trivalent vaccine is used, about 50 such subjects should be examined, or even more if other interfering viruses are known to be present in the community. The sample must be selected on sound statistical principles.

The campaign last year was marked by predominant emphasis on the school-going population, i.e. children from 6 years upwards. Special provision was made for their immunization at school. This resulted inevitably in a gap in the protection of pre-school children, creating a hazardous hiatus in the community, as we pointed out last year.[5] Unless all children from 3–6 months upwards are immunized as well, it will never be possible to produce a highly protected community, with the catastrophic risks which this must entail for the

3. WHO Expert Committee on Poliomyelitis (1960): *Third Report*, p. 11. Geneva.

4. *Ibid.*, p. 15.

5. Editorial (1960): Med. Proc., **6**, 570*a*.

Die onvermydelike gevolg hiervan was 'n leemte in die beskerming van voorskoolse kinders, waardeur 'n gevaarlike hiaat in die gemeenskap geskep is, soos ons verlede jaar daarop gewys het.[5] Tensy alle kinders tussen 3–6 maande en ouer ook geïmmuniseer word, sal dit nooit moontlik wees om 'n doeltreffend beskermde gemeenskap tot stand te bring nie—met al die katastrofiese gevare wat dit noodwendig meebring vir diegene wat aan die kwaadaardige, wilde soorte blootgestel word.

Om maksimum-welslae te behaal met die tweede fase van die veldtog (aangekondig vir Mei van vandeesjaar) moet alle moontlike stappe gedoen word om ook die voorskoolse bevolking in te sluit. As voldoende aandag bestee word aan die talle kleuterskole dwarsdeur die land waar 'n groot ‚gevange' bevolking van hoogs vatbare kinders beskikbaar is, behoort die taak aansienlik makliker te wees.

Sekere voorsorgsmaatreëls moet tydens die veldtog getref word. Die Komitee van Deskundiges op die gebied van poliomiëlitis, aangestel deur die Wêreldgesondheidsorganisasie, het in sy *Derde Verslag* aanbeveel dat sorg aan die dag gelê moet word by die toediening van lewende entstof aan persone wat met kortikosteroïede behandel word, of orofarinksoperasies ondergaan, veral in die geval van drievoudig negatiewe volwassenes. Elektiewe prosedures, insluitende mangel- en adenoïeduitsnydings, moet nie onderneem word solank 'n grootskeepse mondelinge poliovirusvaksinasie-veldtog aan die gang is nie; dit geld veral vir persone wat die mondelinge vaksien onlangs ontvang het.

Die betreurenswaardige en miskien heeltemal dwase wenk is onlangs aan die hand gedoen dat poliovaksinasie verpligtend gemaak behoort te word. In beginsel is dit stuitend. Dit is ook onnodig, heeltemal afgesien van die feit dat so 'n voorstel nie gedoen word wat ander dodeliker siektes betref nie. Temeer, daar is geen wetenskaplike basis vir so 'n maklike toevlug tot dwangmaatreëls nie, aangesien geen poliovaksien (parenteraal of mondeling) 100% onvatbaarheid kan verseker nie. Dit is veel meer demokraties en trouens ook verstandiger om die publiek behoorlik in te lig, en die gewetes van ouers tot volledige en vrywillige samewerking aan te wakker.

Ons is seker dat indien 'n doeltreffende opvoedkundige veldtog betyds aangepak word, die steun van die publiek (wat noodsaaklik vir welslae is) sonder enige moeite verkry sal word. Maar *pari passu* hiermee is dit noodsaaklik om ook die mediese professie betyds in te lig oor al die wetenskaplike aspekte en implikasies van die entstof, insluitende bewyse van die veiligheid en doeltreffendheid daarvan, en die gevare (indien enige) voortspruitende uit simiaanse virusse as verontreinigers tydens weefselkweking. Die Komitee van Deskundiges insake Poliomiëlitis, aangestel deur die Wêreldgesondheidsorganisasie, sê in sy *Derde Verslag* op bladsy 37:

‚Hoewel die meeste simiaanse middels onskadelik vir die mens is, kan daar nie sonder meer aangeneem word dat almal onskadelik is nie, en alle moontlike pogings behoort in die werk gestel te word om hulle uit die vaksien te elimineer.'

Slegs deur almal wat by die saak betrokke is volledig in sy vertroue te neem, kan die owerheid die hoeksteen lê van 'n doeltreffende en geslaagde veldtog om poliomiëlitis uit te roei.

5. Redaksioneel (1960): Med. Bydr., **6**, 570a.

unprotected as a result of exposure to the virulent, wild strains.

In the second phase of the campaign (announced for May this year), every care should be taken to embrace the pre-school population, so as to obtain maximum success. Adequate attention to the numerous nursery schools throughout the country, where a large captive population of highly susceptible children is available, should simplify this task considerably.

Certain precautions must be taken during a campaign. The WHO Expert Committee on Poliomyelitis recommended in its *Third Report* that care should be taken in administering the live vaccine to persons undergoing treatment with corticosteroids or surgery of the oropharynx, particularly in the case of triply negative adults. Elective procedures, including tonsillectomies and adenoidectomies, should not be undertaken when a mass oral poliovirus vaccination programme is in progress, particularly with regard to subjects who have recently received oral vaccine.

The deplorable, if not fatuous, suggestion has recently been put forward to make polio vaccination compulsory. This is repugnant in principle. It is also unnecessary, quite apart from the fact that the proposal has not been made for other more lethal diseases. There is, moreover, no scientific basis for such a ready resort to authoritarianism, since no polio vaccine (parenteral or oral) has been shown to produce 100% immunity. It is far wiser to follow the more democratic (and sensible) procedure of enlightening the public and stirring the conscience of parents to full and voluntary co-operation.

We feel sure that if an adequate campaign of education is undertaken in good time, support on the part of the public (which is essential for success) will be won very readily. But *pari passu* with this it is essential to inform the medical profession timeously about all the scientific aspects and implications of the vaccine, including the evidence of its safety and efficacy, and the risks (if any) from simian viruses as contaminants during tissue culture. The WHO Expert Committee on Poliomyelitis states in its *Third Report* (p. 37):

'Although most simian agents are probably harmless to man, it cannot be presumed that they all are, and every effort should be made to eliminate them from the vaccine.'

Only by taking everyone concerned fully into their confidence, will the authorities lay the foundations for an effective and successful campaign to eradicate poliomyelitis.

VOGT-KOYANAGI-HARADA SYNDROME

UVEO-ENCEPHALITIS

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This syndrome consists essentially of bilateral uveitis associated with widespread disturbances involving the skin, hair, hearing and often cerebral disturbances. It has long been recognized by ophthalmologists.

Reed *et al.*,[1] in an excellent review, pointed out that this condition may present a diagnostic problem to physicians, neurologists, neuro-surgeons and dermatologists. They describe, *inter alia*, one case where exploratory craniotomy was performed in an attempt at diagnosis.

Gregory[2] described a case where the initial symptoms pointed to a diagnosis of intracranial tumour. The patient was referred from Moorfields Eye Hospital to the National Hospital for Nervous Diseases, where she first developed the associated eye symptoms.

Duke-Elder[3] has suggested that the condition appears to be quite common in Japan; a review of the literature indicates that it is commoner in Japan than elsewhere.

It is our belief that this condition (described as relatively rare) is probably not as rare as would appear and that isolated cases occur among the coloured races in South Africa, but do not make their appearance in print. We have knowledge of several cases which were not reported. It is for this reason that we report the following case, which is particularly interesting as the early signs pointed to a cerebral condition and only later did the diagnosis become apparent.

CASE HISTORY AND FINDINGS

A Coloured female, aged 50, was admitted in a confused state to another hospital on 30 March 1960. No history was obtainable.

Examination revealed a mal-nourished, confused patient. Both eyes deviated to the right with maintained nystagmoid movements. Blood pressure: 105/70 mm. Hg. Temperature: 99° F. Pulse rate: 85 per minute. It was noted that 'the sclerae were injected' and the pupillary reactions were doubtful. There was increased tone on her left side and all jerks on that side were increased. The plantar reactions were flexor.

Blood Picture: Hb: 16.6 g. %.
White Blood Cells: 8,600 per c.mm. (no differential count available).
E.S.R.: 25 mm. in 1 hour.
Wassermann Reaction: Negative.
Cerebrospinal Fluid: Pressure: 150 mm. H_2O.
Polymorphs: 4 per c.mm.
Lymphocytes: 24 per c.mm.
Red Cells: 575 per c.mm. (bloody tap).
Protein: 43 mg. per 100 ml.
Chlorides: 615 mg. per 100 ml.
Glucose: 71 mg. per 100 ml.
Blood Chemistry: Blood Urea: 23 mg. per 100 ml.
Chlorides: 103 mEq. per litre.
Sodium: 129 mEq. per litre.
Potassium: 4.1 mEq. per litre.
Urine: Nil abnormal detected.
X-rays: Skull and chest: Normal.

An admission diagnosis of cerebral thrombosis was made. The next day she was seen by an ophthalmologist who diagnosed iridocyclitis and prescribed Atropine and Chloromycetin as well as subconjunctival Mydricaine injections.

During her stay at the hospital she ran an intermittent, mild pyrexia with temperatures averaging 99° to 100° F., and rising to 101° F. on one occasion. Systemic treatment (penicillin 500,000 units *b.d.* and nicotinamide *b.d.*) was also given.

On 18 May 1960 she was transferred to the Baragwanath Hospital Eye Unit. The patient was still confused and appeared to be deaf.

Clinical Findings:
Vision: Right eye: Light perception.
Left eye: Light perception.
Roving nystagmus.
Bilateral iridocyclitis with posterior synechiae and bound-down pupils.
Tension to digital palpation: soft.
Bilateral lens changes; fundi not visualized.

General examination revealed 'shark skin changes' of the extremities, and hepatomegaly. The deep tendon reflexes were increased on the left.

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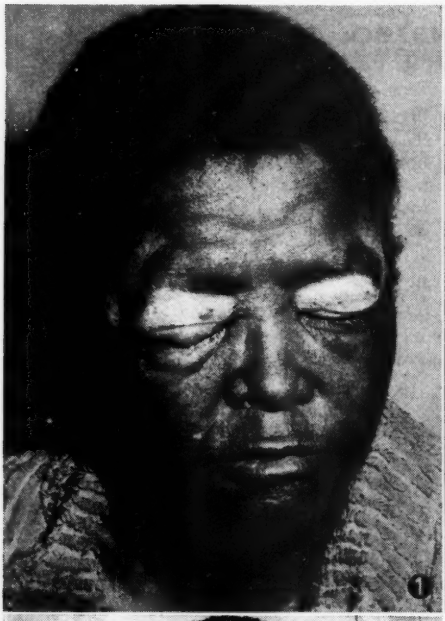
Blood Examination: Hb: 15.4 g. %
E.S.R.: 31 mm. in 1 hour.
White Blood Cells: 8,000 per c.mm.
Segmented Neutrophils: 45%.
Monocytes: 9%.
Lymphocytes: 33%.
Eosinophils: 13% (absolute eosinophilia).
Platelets: Adequate.
Wassermann Reaction: Negative.
Urine: Nil abnormal detected.
Cerebrospinal Fluid: Clear, colourless, pressure normal.
Lymphocytes: 6 per c.mm.
Protein: 10 mg. per 100 ml.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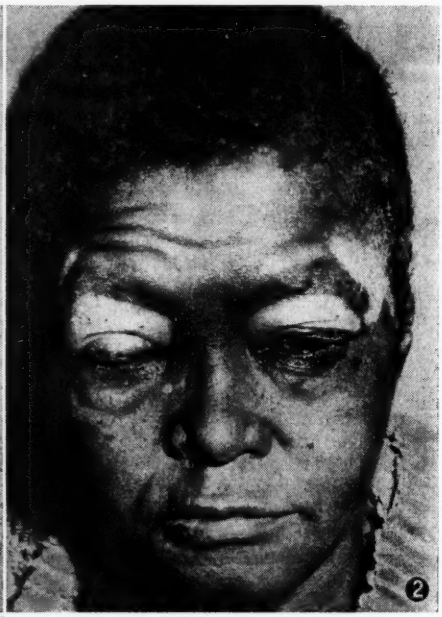
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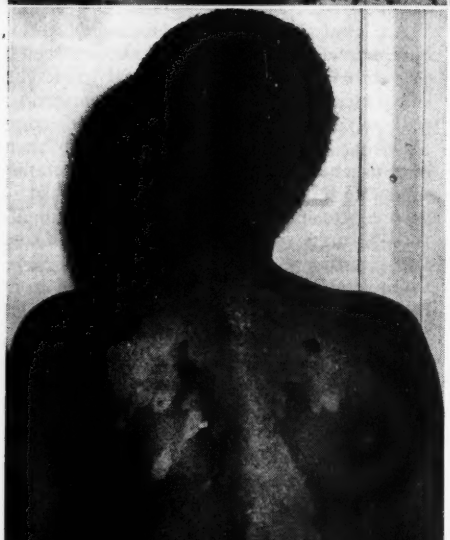
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Sugar: 90 mg. per 100 ml.
Sodium Chloride: 640 mg. per 100 ml.
Lange's Colloidal Gold Test: Negative.
Mantoux Test: Negative.
Toxoplasmosis Test: Negative.
Histoplasmosis Test: Negative.
X-rays: Chest, skull, sinuses: Normal.

The patient was seen by an ear, nose and throat specialist who diagnosed nerve deafness.

The association of mental confusion, deafness and iridocyclitis suggested the diagnosis of the Vogt-Koyanagi-Harada syndrome, and 13 days after admission it was noticed that the skin over the temples had turned white (vitiligo), the hair line was receding (alopecia) and a whitening of eye lashes, eye brows and hair (poliosis) was occurring (Figs. 1 and 2). The vitiligo involved the eye lids, temples and also her back (Fig. 3). The condition remained unchanged.

In this case the original diagnosis was cerebral disease but the correct diagnosis later became obvious. Figs. 1 and 2 show the poliosis (whitening of the hair) and the vitiligo (patchy depigmentation of the skin) particularly well and the diagnosis is obvious here; but the

importance of this case to all clinicians consists in the early signs of mental confusion associated with iridocyclitis. It can be easily understood how such patients can go on to unnecessary neuro-surgery.

SUMMARY

A case of Vogt-Koyanagi-Harada syndrome is reported, showing the signs of mental confusion, iridocyclitis, deafness, poliosis, vitiligo and alopecia.

We should like to thank Dr. I. Frack, Superintendent, Baragwanath Hospital for permission to submit this case for publication.

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SYMPATHETIC OPHTHALMIA OR THE VOGT-KOYANAGI-HARADA SYNDROME?

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It has long been suggested that there is a relationship between sympathetic ophthalmia and the Vogt-Koyanagi-Harada syndrome, and the reason for reporting this case is two-fold: firstly, to draw attention to the relationship and secondly to present some facets of the different and unexpected response of the eyes of Bantu patients to trauma.

Reed *et al.*[1] in their excellent review, draw attention to the relationship which has been suggested between these two conditions and reference to Duke-Elder[2] confirms the similarities between the two conditions as they affect the eye.

CASE REPORT

A Zulu female, aged 24, was admitted on 23 March 1960 to the Baragwanath Eye Unit, complaining of pain and poor vision in her right eye for 7 months before admission. Following trauma, her left eye had been removed during August 1959 at another hospital. One week after discharge from that hospital, she stated that her right eye became painful and her vision started deteriorating.

Vision: Right eye: Hand movements at 2 metres. Ciliary congestion.

Numerous pigmented K.P. and flare in anterior chamber, with posterior synechiae and lens opaque.

There was patchy depigmentation of the skin and the hair on the forehead.

General Examination: No pyrexia. Blood pressure: 120/80 mm. Hg. Pulse rate: 84 per minute.

Physical: No abnormalities demonstrated. No deafness.

X-rays: Skull and chest: no abnormalities detected.

Blood Picture:

Hb: 15 g. %.

White Blood Cells: 7,200 per c.mm. (normal differential count).

E.S.R.: 18 mm. in 1 hour.

Urine: No abnormalities detected.

Wassermann Reaction: Negative.

Cerebrospinal Fluid: No abnormalities detected.

Investigations were negative for tuberculosis, toxoplasmosis, sarcoidosis and brucellosis.

Treatment. Local treatment for the iridocyclitis was instituted plus high dosage steroids.

She lost her anterior chamber one month after treatment commenced, with a rise in tension which was controlled with Diamox, fluctuated and then settled after a month. As vision had deteriorated to light perception only, the lens was extracted after freeing adhesions, but sight was not improved. The fundus could not be seen owing to the hazy vitreous.

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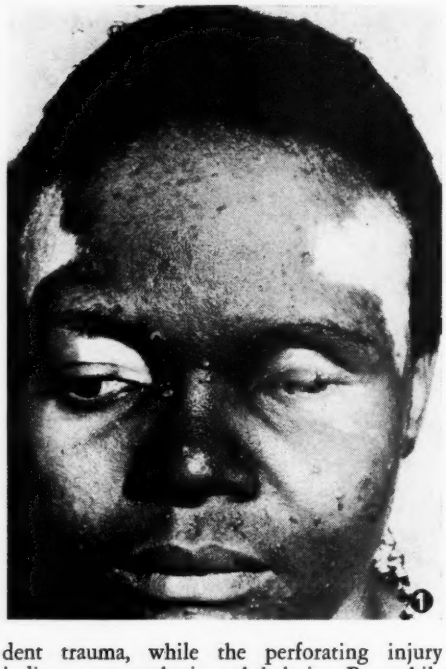
DISCUSSION

This patient presents features (Fig. 1) which would allow classification in either category namely, the poliosis and vitiligo indicate the Vogt-Koyanagi-Harada syndrome with coinci-



dent trauma, while the perforating injury indicates sympathetic ophthalmia. But while the Vogt-Koyanagi-Harada syndrome occurs occasionally in the non-White patient (Trope and Coetzee[3]) sympathetic ophthalmia apparently does not.

With the possible exception of this case we have found that no cases of sympathetic ophthalmia have been reported in the Bantu, in spite of the large number of patients treated every year for perforating eye injuries.

At Baragwanath Hospital the number of patients treated for direct injuries to the eyes (and this excludes all cases where eye injuries are coincidental to other injuries) is of the order of 25 per month; yet in the last 10 years no case has been diagnosed as sympathetic ophthalmia.

Personal inquiries from older colleagues who have treated Bantu patients in large numbers elicit the same response and we know that, with the exception of one or two doubtful cases, no South African ophthalmologist in the Johannesburg area has seen a case which has convinced him beyond reasonable doubt to be sympathetic ophthalmia.

This does not apply to the White population, or to the Coloured population, where the occurrence of sympathetic ophthalmia has been noted in both groups.

The question is raised: why does sympathetic ophthalmia not occur in the Bantu? We do know that the uveal tract is involved in these conditions and we suggest that the excess of uveal pigment in the Bantu may impede transmission of the causative factor when induced by trauma. We have noted that iris pigment is liberated in relatively large quantities during lens surgery and when alpha chymotrypsin has been used the iris often tends to stick to the lens and must be freed before delivery. Detached retina is also not common. When it does occur there is often spontaneous cure, so much so that the prognosis in operated cases is very good. These differences, viz. in the effect of alpha-chymotrypsin on the iris and the prognosis in retinal surgery are, in our opinion, possibly associated with the fact that sympathetic ophthalmia does not occur in the Bantu.

It is not merely the presence of pigment in large quantities that plays this part, as Coloured and Indian patients react differently from Bantu patients in this respect. It is probably either pigment in much larger quantities or of different quality, possibly irritative, that seems to produce rapid healing in detachments and prevents sympathetic ophthalmia.

SUMMARY

A case (?) sympathetic ophthalmia or (?) Vogt-Koyanagi-Harada syndrome is described.

Attention is drawn to the relationship of these two conditions.

Comment is made on the non-occurrence of sympathetic ophthalmia in the Bantu patient and it is suggested that uveal pigment plays a part in this immunity.

We should like to thank Dr. I. Frack, Superintendent, Baragwanath Hospital for permission to submit this case report for publication.

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ORTHODYSARTHRICS

THE NEUROCEPHALALGIAS

A RE-APPRAISAL AND SUGGESTED AETIOLOGY

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'Observed facts must always take precedence over theory.'—*White & Sweet (1955).*

Whether presenting alone or as part of a complex syndrome, headache is among the most demanding conditions which daily face the general practitioner. In its more complex manifestations, diagnosis and treatment necessitate the exhibition of considerable patience and clinical acumen.

A cephalic discomfort is inevitably more disconcerting to the patient than a physically comparable discomfort in any other part of the body. In headache (a psycho-somato-autonomic reaction to a noxious stimulus) the somatic component (pain) is frequently overshadowed by undue emphasis on the psychic element, with the consequent danger of postulation of a psychogenic aetiology, inadequate attention being directed to the essentially physical causation.

The frequent application of the terms 'idiopathic' and 'functional' to various types of headache is illustrative of this danger.

'Once the diagnosis of migraine . . . is established, almost all cases are "idiopathic" unless they belong to the group occurring later in life. . . . Migraine is surely the extreme example of a malady in which the degree of suffering depends on the sufferer. . . .' (Heaf, 1959).

Whether such a condition as psychogenic headache (i.e. a headache which is *primarily* psychogenic) exists at all, is open to question; but there is no question about the impropriety of labelling a condition as being of such origin until all other possibilities have been eliminated. The use of the term 'idiopathic' is no more than a confession of ignorance. *A syndrome occurs, therefore it must have a primary physical cause.* A search must be made for this cause.

The 'idiopathic' attitude is particularly prevalent in the field of the neuro-cephalalgias. It is the purpose of this paper to record certain observations made in this field in the hope that they may offer a possible clue to the causation of certain syndromes, of which headache, whether it takes the form of neuralgia, cephalalgia or a combination of the two, is the predominant symptom.

From the point of view of present treatment, 'headaches' can be divided into 2 main classes:

(*a*) Those in which treatment is directed at the removal of the cause. This presupposes that the cause is known and is accessible to treatment.

(*b*) Those conditions for which treatment is symptomatic. Such treatment is applied where the cause is:

i. Inaccessible.
ii. Not amenable to any known treatment.
iii. Not known.

The types of neurocephalalgia which will be discussed are to be found in (*b*) iii. They include:

i. Idiopathic trigeminal neuralgia.
ii. Atypical facial neuralgia.
iii. Migraine.
iv. Autonomic facio-cephalalgia.

This classification is based on that of White and Sweet, 1955.

Historically, these syndromes were customarily described under the inclusive term 'hemicrania.' It is only comparatively recently that subdivisions have been created. This symptomatic subdivision implies a differing aetiology. In spite of this, it is interesting to note that most authors, however much they endeavour to emphasize the particularity of the syndrome which they happen to be discussing, do admit the existence of intermediate forms.

As various theories have been advanced for the mechanism of causation of these syndromes, so have treatments been evolved to deal with these hypothetical mechanisms. The accepted treatment of trigeminal and facial neuralgia is interference with the continuity of the Vth nerve, either by surgery or by injections of alcohol. Migraine and autonomic facio-cephalalgia are now treated by an attack on the vasomotor mechanism of the vessels in the affected area, either surgically or by ergotamine compounds.

But (and this is a point of fundamental importance) *all the treatments in customary use are, in fact, purely symptomatic.* They deal only with the mechanism of transmission

of symptom-producing impulses, or the pathways of conduction of the impulses producing the symptoms. They do not deal with the *primary, precipitating cause* of the syndromes. This cause has, as yet, not been identified in any of the conditions under discusion.

It is unnecessary here to discuss the detailed symptomatology of the neurocephalalgias; the reader is referred to the study of these conditions by White and Sweet (1955) and to papers on individual conditions by the various authors in the list of references.

The characteristic symptomatology of the various syndromes, when extracted from the works of these authors, may be summarized as in Table 1.

TABLE 1

A. Idiopathic Trigeminal Neuralgia.
B. Atypical Facial Neuralgia.
C. Migraine.
D. Autonomic Facio-cephalalgia.

	A.	B.	C.	D.
1. Paroxysmal	+	+	+	+
2. Cyclical	+	+	+	+
3. Severe	+	+	+	+
4. Unilateral	+	+	+	+
5. Sensory changes	+	+	+	+
6. Trigger points	+	+	+	+
7. Neuralgia	+	+	+	+
8. Cephalalgia	−	+	+	+
9. Nausea	−	±	+	+
10. Vomiting	−	−	+	+
11. Histamine precipitated	−	−	±	±
12. Alcohol	−	−	+	+
13. Neurotic personalities	−	−	+	+
14. Autonomic symptoms	−	−	+	+
Vaso-dilation	−	−	−	+
Lacrimation	−	−	−	+
Nasal congestion	−	−	−	+
Conjunctival injection	−	−	−	+

There is a suggestion here that at least in the order shown, each of these conditions is apparently a more complex manifestation of the previously mentioned syndrome. Van Storch, quoted by Friedman and Mikropoulus (1958) found that histamine produced headache in migrainous subjects, sufferers from atypical facial neuralgia and non-migrainous subjects, in that descending order of frequency. He also found the 2 former to be more sensitive to histamine *during the times when they were suffering from bouts* of their own particular type of neurocephalalgia. This finding would suggest that, at these times, some mechanism was operative which caused the patient to be in a 'state of sensitivity', and that only the application of some further specific stimulus was necessary to produce a characteristic attack of their particular disability.

If the basic similarity of the syndromes, and the concomitant existence of a 'state of sensitivity, are taken into account, it would not appear unreasonable to suspect a common cause for the precipitation of the 'state of sensitivity' and the subsequent development of an attack or bout of attacks.

The identification of such a common cause can only come from clinical observations made during an intimate study of individual cases of the conditions concerned. The clues pointing to this common cause are most likely to be found immediately before, during and after an attack or bout of attacks, in other words, during the 'state of sensitivity,' the *'status neurocephalalgicus.'* The general practitioner is the person most likely to be in the privileged position of observer at this significant time.

During the past year I have encountered 32 cases of neurocephalagia of various types. These cases can be allocated to the various subdivisions as follows:

TABLE 2

'Idiopathic' trigeminal neuralgia (1–4)	4 cases
Atypical facial neuralgia (5–17)	13 cases
Migraine (18–26)	9 cases
Autonomic facio-cephalalgia (27–32)	6 cases
Total	32 cases

TYPICAL CASES FROM EACH SUBDIVISION

Space precludes the reporting of full histories of all cases upon which the hypothesis presented in this paper is based. Abbreviated summaries of selected cases will therefore be given with comment on cases considered to be of particular significance.

COMMENT ON TABLE 3

Case 1. Suffered from 'migraine' for years; relieved by chiropracter. In 1954 dental extractions were followed by onset of facial pain of burning character followed by a sharp pain occurring in increasingly frequent bouts. Trigeminal neurectomy in 1958. Only four days freedom. Attacks were so severe subsequently that she had to be admitted to a nursing home where she was kept under Pethidine (two occasions). A stellate ganglion block eliminated the sharp pain for some hours, paraspinal block eliminated the burning pain except for 'pins and needles' in left upper lip. Combined blocks repeated relieved both pains for up to twenty-four hours. Meanwhile a course of general manipulation plus direct to T 7–9 was followed by a decrease in the intensity of the pain to the extent that she put on

6 lb. in weight. Reported 5 months after end of treatment that she was almost completely free from pain.

Case 2. An African female, was *immediately* relieved of a very severe attack. No follow up.

Case 3. Had over a period of 27 years been diagnosed as 'sinusitis'. She has been subjected to frontal sinusectomy and maxillary antrostomy. A tooth root remnant on the affected side was considered to be a possible cause of her symptoms but

TABLE 3: IDIOPATHIC TRIGEMINAL NEURALGIA

No.	*Complaint*	*Duration*	*Type of Pain*	*Side*	*Spinal Component*	*Hyperaesthesia*	*Treatment*	*Result*
1.	Pain in brow, eye, cheek, lip	6 years	Burning, plus sharp	Left	T8-9	TGN Trapezius T8-9	Cervical thoracic manipulation. S.G.B. Para-spinal Block	Considerable relief
2.	Pain in eye, brow, cheek	Periodically for some years	Sharp, burning	Right	T7-8	T7-8 Trapezius TGN I-II	Thoracic manipulation T7-8	Immediate relief present attack
5.	Constant sharp pain	27 years	Sharp	Right	T8-9	TGN I-II Trapezius T8-9 R	Direct T7-9	Immediate relief 8 months
4.	Pain in brow, eye, cheek	8 years	Sharp, burning	Left	T9-10	T9-10 Trapezius	Left district before treatment could be carried out	

TABLE 4: ATYPICAL FACIAL NEURALGIA

No.	*Complaint*	*Duration*	*Type of Pain*	*Side*	*Spinal Component*	*Hyperaesthesia*	*Treatment*	*Result*
5.	Pain in left eye with frontal ache	4 days	Shooting	Left	T8-9	T8-9 TGN I Trapezius	Ceiling traction T8-9 manip.	Immediate relief
7.	Right sided headache	2 days	Dull ache	Right	T6-7	T6 TGN I, II Trapezius	Direct T6-7	Immediate relief
9.	Pins and needles shoulders. Attacks of pain side of head and face. 'Pricking' T9	1 week	Sharp	Left	T9	T9 TGN I, II Trapezius	Seated rotation R.	Relief
10.	Pain in eye and frontal	3 days	Shooting	Left	T8	T8 TGN I Trapezius	Direct	'Headache gone'
12.	Pain side of face. Vertigo	2 days	Constant	Left	T8	T8 TGN II Trapezius	Direct	All symptoms relieved
13.	Headache, pain neck and throat	2 days	Dull	Left	T8	T8 TGN III Trapezius	Direct	Recurred twice after 24 hours. Treatment repeated—free
16.	Headache over right eye	1 year	Periodic, more frequent	Right	T8	T8 TGN I Trapezius	Direct	'That feels better Doctor.'

its removal gave no relief after three weeks. Direct thoracic manipulation was performed on 16.1.60. On 19.1.60 she reported complete freedom from pain. On 2.8.60 she stated that she had only suffered occasional twinges since January 1960.

COMMENT ON TABLE 4

These cases are selected as being representative of the others in this category.

Case 9. Has had two previous instances of dysarthria, both cervical. This was the first occasion on which a thoracic component was found, though its significance remained unrecognized.

Case 12. Was complicated by a severe vertigo which was particularly noticeable when lying down or turning over. The speed of relief was remarkable.

Case 13. The pain was extremely severe. He admitted to previous similar attacks but none of this severity. The recurrences occurred on this occasion shortly after rising from bed. On each occasion manipulation produced dramatic relief.

Case 16. A child of 9 who had had a cycle accident a year previously. He had been unconscious at that time for 24 hours. The attacks started subsequently and were becoming increasingly frequent. There has been no recurrence (4 months).

COMMENT ON TABLE 5

It is only proposed to discuss two cases in this series as the remainder are essentially similar.

Case 18, *aet.* 43. This is the case which first led to the suspicion that there might be a thoracic component in the aetiology of neurocephalalgia. It will therefore be discussed in some detail.

Mrs. B. M. S. has been inflicted with classical migraine for many years. With the exception of a period of 6 months following a hysterectomy in 1957, she has suffered 2–3 attacks per month, sometimes having 2 attacks in the same week.

The attacks begin with dilatation of the veins of the left arm and hand; this is followed by hyperacusis and photophobia which both become sufficiently intense to be unendurable during the attack which follows. Subsequently there is a temporal ache and hemicrania which, if untreated, passes to nausea and vomiting. In spite of the administration of ergotamine compounds, few attacks have been aborted. Latterly the only effective treatment has been Pethidine and Largactil.

In March 1959, while being treated for an attack of pain in the neck and shoulder by cervical traction and manipulation, she claimed that an attack of migraine had been averted. She had no further attack until July 1959, when she reported with a full-blown attack of migraine with intense pain, photophobia and hyperacusis. She felt nauseous and had to be helped into the surgery.

In view of the previous experience she was immediately subjected to cervical traction. When the pull reached 35 lb. she indicated that the pain was easier. The traction was increased to 55 lb. and maintained for 15 minutes. While traction was being applied an investigation of the thoracic spine was carried out. This revealed a tender area to the left of T 7–8. Knee pressure was applied at this point and a distinct 'clunch' was felt and heard both by patient and operator.

On release of traction the acute pain had disappeared although some discomfort was still present. She still felt 'muzzy', possibly due to tablets of Largactil and Cafergot which she had taken earlier. *Within 20 minutes she was completely free from all symptoms.*

In November 1959 she presented with a partially developed attack which had progressed to hemi-

TABLE 5: MIGRAINE

No.	*Complaint*	*Duration*	*Type of Pain*	*Autonomic Symptoms*	*Spinal Component*	*Hyperaesthesia*	*Treatment*	*Result*
18.	Unilateral headache, left	10 years	Throbbing, sharp	Photophobia, hyperacusis. Nausea, vomiting	T8	Trapezius, TGN I, II T8	Direct T8	Relieved
21.	Unilateral headache, left	Some years	Throbbing	Nausea, vomiting. Photophobia	T8	T8 TGN I, II Trapezius	Direct	Relieved; immediate
22.	Unilateral headache, left	Years, increasing frequency	Throbbing	Hemianoptic blurring of vision, nausea	T7-8	TGN I Trapezius	Direct	Relieved
24.	Unilateral headache, right	20 years increasing frequency	Throbbing and pain	Photophobia, nausea, vomiting	T8	T8 TGN I, II Trapezius	Direct	Relieved
25.	Unilateral headache, left	10 years	Throbbing, acute	Photophobia Hyperacusis	T7-8	T7-8 Trapezius TGN I, II	Direct	Relieved
26.	Unilateral headache, either side	16 years	Throbbing, feels as if eye pushed out	Vertigo, nausea, vomiting	T8-9 Trapezius	T8-9 Trapezius TGN I, II	Direct	Relieved in 2 hours

crania. This was the first incident since July. The attack was aborted by direct manipulation of the T 7–8 region where an area of tenderness and hyperaesthesia was detected.

28 *February* 1960. Impending left sided attack. T 8–9 manipulation produced 'clunk' and the attack did not develop, but trapezius discomfort and a 'headachy' tendency persisted. Subsequent cervical manipulation elicited a 'clunk' at C 6–7. All symptoms disappeared within minutes.

Two impending attacks have been relieved by thoracic manipulation since the above occasion, but no major attack has been experienced since July 1959, a period of thirteen months.

Case 22, *aet.* 43. Has suffered for almost twenty years from attacks of mild migrainous headache. These had become almost daily occurrences. The attacks come on slowly on his first rising in the morning. They are heralded by a blurring of vision of the hemianoptic type. This passes off with the development of the attack. The attack consists of a throbbing ache which may occur on either or both sides.

24 *August* 1959. An attack was present when seen on this occasion. Examination revealed a limitation of neck movements with worsening of the pain on left rotation and hyper-extension. The thoracic region was not investigated on this occasion as the interview occurred in his office. The same afternoon radiology revealed osteo-arthritis of C 5–6. Subsequent traction relieved his symptoms at a pull of 35 lb. A 55 lb. pull was maintained for 15 minutes followed by manual traction and manipulation of the neck. A mild 'clunch' was elicited at C 5–6 (R). He was unable to attend for further treatment until 18 October 1959 when he stated that he had been free from attacks immediately following the previous treatment but they had now recurred, less frequently and less intensely. A full examination was now carried out and this revealed tenderness lateral to T 7–8. Ceiling traction was applied for 15 minutes followed by direct manipulation of T 5–9. A 'clunch' was experienced at T 8. The treatment was repeated on 5 occasions subsequently without any 'clunch' being elicited.

He has had no further attacks to date (August 1960). This case is of interest because it illustrates how an individual will learn to live with a disability if he has been told that nothing can be done for it. It also illustrates how awareness of the possible manifestations of a spondylo-dysarthric lesion can lead to the relief of a considerable amount of unecessary suffering.

COMMENT ON TABLE 6

Case 27, *aet.* 32. Presented with what he claimed was an attack of 'flu' accompanied by a severe headache. He sat holding his head in his hands. T.P.N. Congestion of right eye with lacrimation, congestion of right nostril and flushing of right side of face were present. Gave a history of similar attacks of headache, without the autonomic symptoms but sometimes accompanied by attacks of shivering. He had been told that he had migraine and that nothing could be done about it. The attacks usually lasted about four days and when severe were accompanied by nausea. He never vomited. He was treated by direct traction to the mid-thoracic region, but was not fully investigated on this occasion because of the obvious agony which he was suffering. The manipulation eased the pain and the attack subsided completely in 2 hours (20 October 1959).

17 *November* 1959. He came in for review and said that he had had no further attacks though he felt that one was impending. Examination revealed that he was extremely tender over T 8–9 and pressure over this point caused a pain in the brow and around the eye. A few minutes later he remarked spontaneously, 'Doctor, you've brought on

TABLE 6: AUTONOMIC FACIO-CEPHALALGIA

No.	*Complaint*	*Duration*	*Type of Pain*	*Autonomic Symptoms*	*Spinal Component*	*Hyperaesthesia*	*Treatment*	*Result*
27.	Severe unilateral headache (R)	10 years	Sharp, throbbing	Conjunctival injection. Lacrimation. Nausea	T8-9	T8-9 Trapezius TGN I, II	Direct	Relieved in 2 hours. Normally 3 to 4 days
28.	Acute, severe unilateral headache	7 years in bouts	Excruciating	As above	T8	C5-6 T8 TGN I, II	Various	Relieved
30.	Severe pain neck, face, and mid-back	18 years. Previously migraine	Excruciating	As above. Facial flush	T7-8	T8-9 Trapezius C5-6 TGN I, II	Various	Relieved
31.	'Conjunctivitis', pain in and around eye (1)	3 days	Sharp	Conjunctival injection, nasal congestion. Lacrimation	T5	T5 TGN I Trapezius	Direct	Relieved
32.	Headache, left, pain in eye	3 days	Sharp	Lacrimation, conjunctival injection. Proptosis	T8-9	T8-9 Trapezius TGN I, II	Direct	Relieved

an attack'. In a matter of minutes his appearance was as described above 20 October 1959. A marker was placed over the thoracic tender spot and his spine X-rayed. The tender spot was identified as being over the T 9 costo-transverse process (R). Direct manipulation of this area was accompanied by a sharp 'clunk' which was both felt and heard by the operator. By the time he had dressed the attack had completely subsided.

Contacted by telephone (he had left my district), he stated that he had had only very occasional mild recurrences (7 July 1960).

This case is remarkable for the actual precipitation of an attack and its immediate relief by manipulation.

Case 28. *Aet.* 47. This case was first seen before Case 18 had indicated the possibility of a thoracic dysarthria as the cause of neuro-cephalalgia. He was treated by cervical traction, stellate ganglion block, pethidine and ergotamine compounds on various occasions, with only temporary relief. (March-April 1959).

1 *November* 1959. Having decided to investigate all neurocephalalgias in my records who were still available, I asked this patient to attend for review. He said that he had only had one slight spasm of eye pain since last seen. This had not developed into an attack. He was specifically questioned as to whether he had ever suffered any pain in the thoracic spine during his previous atacks. He admitted that he had so suffered, and that he had had chiropractic treatment for this region following which the attacks had ceased until the previous March. He remarked, 'As a matter of fact I've got it now.' Examination revealed an extremely tender spot 1½ in. lateral to T 8, pressure upon which produced a pain shooting into the suprascapular region, the side of the neck and the eye. Direct manipulation was immediately applied with a 'clunch' at T 8. A few minutes later no pain was produced by pressure over this point. The T 8 dermatome which had previously been *hyper*-aesthesic was now *hypo*-aesthesic. He has had no further attacks (August 1960) but has been successfully treated for paraesthesia of the fingers due to C 7–8 osteo-arthritis. No reference of pain was elicited by pressure over T 8 during the latter course of treatment.

Case 30. 18 *June* 1959. Seen during the night. He was suffering from acute photophobia, and pain, with lacrimation, nasal congestion and flushing of the right side of the face. There was a tender area present to the right of T 7–8 the significance of which was not recognized at the time. He was given pethidine with relief until 7.30 p.m. next day when another attack occurred. A stellate ganglion block gave relief in 7½ minutes. He suffered mild attacks of discomfort until 29 June 1959 when a severe attack occurred. Stellate ganglion block took the edge off but did not relieve completely; however, he ultimately slept and awoke free from pain. Cafergot suppositories were prescribed for use in any further attacks.

27 *July* 1959. He reported several minor attacks apparently controlled by Cafergot. On this occasion, in the middle of a bout, his thoracic spine was investigated. Pressure over the right T 7 costo-transverse joint caused a stab of pain in and around the eye, injection of the conjunctiva and pain in the neck, arm and supra-scapular area. Direct manipulation of T 6–8 was followed by a 'clunch' under the operator's hand. All symptoms disappeared within a few minutes. Subsequent pressure over the rib-head caused no pain and no tenderness was present.

8 *August* 1959. Reported complete freedom from attacks.

Case 31. *Aet.* 10. Presented with an acute attack of *unilateral* conjunctivitis which the mother suggested was due to excessive chemicals in a swimming pool.

10 *September* 1959. The 'conjunctivitis' was accompanied by lacrimation, nasal congestion and pain in the left eye and forehead. Tenderness and hyperaesthesia were present to the left of T 5–6. Pressure over this point increased the eye pain. Direct manipulation relieved all symptoms within minutes.

28 *November* 1959. Recurrence of above without lacrimation. T 5–6 tender. Direct manipulation relieved.

1 *August* 1960. No further attacks.

Case 32. *Aet.* 42. 13 *May* 1960. Father of Case 31. Left sided headache with pain in the left eye and some degree of ptosis and conjunctival injection. Neck movements limited to left. Manual traction and manipulation of neck with 'clunch' at C 5–6.

14 *May* 1960. No change. Area of tenderness to *right* of T 8–9. Direct manipulation relieved immediately.

20 *May* 1960. Slight recurrence. Tender T 8–9. Manipulation relieved. No recurrence.

DISCUSSION

All but 4 of the cases included in this study have occurred in one general practice. Four (Cases 1, 2, 19 and 24) have been referred by colleagues.

From the observations made in these 32 cases certain conclusions can be drawn:

(*a*) They are all eligible for inclusion in one or other of the categories postulated by White and Sweet (1955). The 32 cases include all cases of neurocephalalgia encountered by the author during the period March 1959—July 1960. They have been specifically differentiated from the other types of 'headache' such as that due to hypertension, sinusitis, intracranial pathology, etc. and from local conditions of the head and neck, often described as 'headache' by the patient, but which are, in fact, of purely cervical origin and normally consist of single incidents of acute onset rather than recurrences of a particular syndrome.

(*b*) Though each case has been allocated, for the purpose of discussion, to one or other of these categories, according to its symptomatology, with the exception of the 'idiopathic' trigeminal neuralgias, there is considerable overlapping, as some cases could, on superficial analysis, qualify for a category other than that in which it has been placed.

This overlapping, or existence of intermediate types, becomes increasingly evident as the

history and symptomatology are ascertained in greater detail. In general practice, at least, there is a tendency to label every case of recurrent 'headache' as being 'migraine.' Provided the bouts or attacks remain relatively infrequent and mild, it is left at that. It is only when the attacks become frequent and severe that an effort is made to investigate them more fully with a view to applying alternative methods of treatment.

It is also of interest that the patients in Classes I, II and IV have themselves recognized that the nature of their particular syndromes varies from bout to bout or that they have previously suffered from attacks of a different type, e.g. Cases 1, 27, 30.

(*c*) *All the cases under discussion exhibit a thoracic component, in that a lower thoracic subjective signs precede and accompany the acute attacks.*

(*d*) A 'condition of sensitivity' occurs in these patients which is only present before and during the single incidents or bouts of attacks. It is on this 'state of sensitivity' (status neurocephalalgicus) that the acute attacks are superimposed.

The latter two findings are of the utmost importance in the elucidation of the aetiology of the neurocephalalgias. The finding of the thoracic component of local tenderness and referred hyperaesthesia, and the consequent implication that an intervertebral joint disorganization underlies the syndromes, suggests the possibility of relief by means of appropriate manipulative treatment.

The existence of the 'state of sensitivity' preceding and accompanying attacks and its absence between bouts and in non-neurocephalalgic individuals, was most marked. During the 3-month period December 1959—February 1960, every patient attending the author's surgery was examined for the presence of a tender area in the lower thoracic spine. In no instance, except in patients suffering from neurocephalalgia and those presenting other dysarthric manifestations, e.g. intercostal neuralgia, 'backache' or, on occasions, abdominal symptoms, was such an area of tenderness elicited.

The precipitation of a typical attack during a 'state of sensitivity' by pressure over the tender area is considered as providing confirmation that the intervertebral joint complex is implicated in the production of symptoms, especially when the attack so precipitated can be immediately relieved by the appropriate manipulation. Cases 1, 4, 27 and 30 reacted to finger pressure in this way, while Cases 3, 13, 20, 21 and 27 suffered an increase in facial pain when the tender area was pressed upon, or when, with the patient in the prone position, the leg on the affected side was raised posteriorly.

This observation appears to indicate that a specific neurological linkage exists between the lower thoracic region and the trigeminal distribution. The existence of an area of tenderness in the trapezius and the production of pain in this area by pressure on the thoracic tender area or posterior leg-raising, would suggest a similar linkage. The nature of this linkage will be discussed later.

(*e*) There is no doubt that suitable manipulation of the affected thoracic area results in the reduction of an intervertebral joint disorganization. The 'clunk' felt and heard by both patient and operator is precisely similar to that experienced on the reduction of any other joint. Radiology, with a marker over the tender area, pin-pointed it as lying over the costotransverse joint. This latter observation is not complete proof of the involvement of any particular joint, but taken in conjunction with the known innervation of the paraspinal joints (the posterior ramus) it is, at least, circumstancial evidence.

ANATOMICAL AND NEUROLOGICAL CONSIDERATIONS

It has been suggested elsewhere (Dalgleish, 1959) that disorganization of the intervertebral joint complex is responsible for the various syndromes which are amenable to manipulation. It was further suggested that the diverse manifestations of this disorganization, dermatomal, myotomal, scleratomal and autonomic, are instigated by pathological or traumatic changes in the components of the intervertebral canals which, by pressure, angulation or spread of inflammation, initiate impulses in the nerve structures in, and related to, them.

After this suggestion was put forward, Prof. E. N. Keen, of the University of Natal, drew the author's attention to the works of Pedersen *et al.* and of Stilwell (1956). The former in man, and the latter in monkeys, have described the innervation of the paraspinal intervertebral joints. The former identified twigs from the *posterior ramus* as supplying these joints and the latter described in detail the existence of a *plexus* lying in the intervertebral canal which contains elements from the spinal nerves, the rami communicantes and the sympathetic chain. He demonstrated further that each plexus supplied not only its own but also the

subjacent complex. Incidentally, no evidence of the innervation of the intervertebral joint complex has found its way into the textbooks. It would appear that the importance of these structures has been overlooked.

The complexity of Stilwell's plexus (Figs. 1A, 1B) and the relatively widespread distribution of its branches, both afferent and efferent, explains the varied nature of the manifestations of traumatic and pathological changes in its environment. These manifestations may be further complicated by the existence of the 'branched axon' (Sinclair, 1948) in the nerve trunks concerned.

warning and reparative systems of the body, the greater sensitivity demonstrated by these authors is not unexpected. At what point in the body are they more exposed to environmental changes of a pathological nature than in the intervertebral canals?

The acceptance of the hypothesis that complex syndromes may be initiated by intervertebral joint disorganization, and the evidence that correction of this disorganization, by suitable manipulative procedures, does resolve the syndromes, requires a re-orientation of the present attitude towards all syndromes having dermatomal, myotomal, scleratomal and/or

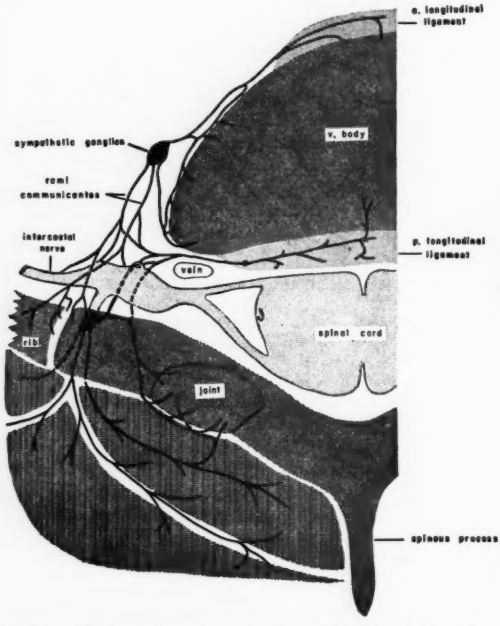

Fig. 1A. Horizontal section at a thoracic level showing the main branches of the spinal ganglion and of the dorsal ramus. A paravertebral nerve plexus is shown (see text), made up of communications between the spinal and sympathetic ganglia, with branches to the longitudinal ligaments, dorsal and ventral (intercostal) rami, periosteum, spongy bone of the rib, the vertebral body and arch, and to costotransverse and intervertebral joints. The sources of the meningeal ramus ('recurrent' nerve) are shown. Muscular branches of the dorsal ramus give off nerves to the intervertebral joints; these correspond to articular nerves in figure 2.

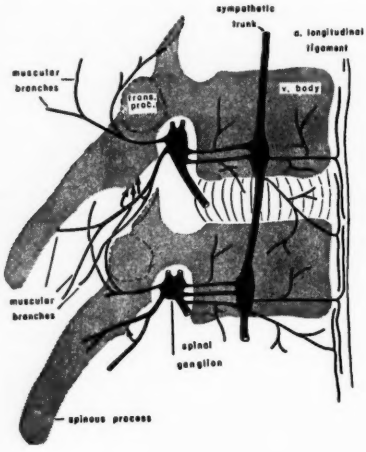

Fig. 1B. Dorsal and ventral ramus branches in the thoracic region. A cranially directed branch of the dorsal ramus supplies deep oblique muscles and an articular capsule cranial to its level of origin. Another dorsal ramus branch supplies a joint one segment caudal, continuing into dorsal muscles. Ventral rami (not labelled) have been severed. Autonomic rami, two or more in number, give off branches to periosteum and areolar connective tissue on the surface of the intervertebral disk. The anterior longitudinal ligament and nearby periosteum receive nerves which arise from the sympathetic ganglia.

It has been shown by Sunderland and Ray (1945–48) that sensory and autonomic fibres are more susceptible to injury than are motor fibres. Maher (1960) has demonstrated the selective action of phenol on sensory fibres, both A and C. As these fibres are part of the vascular (autonomic) components. In considering the possible variants of the manifestations of intervertebral joint disorganization (spondylodysarthria), the existence and complexity of Stilwell's plexus, the existence of the 'branched axon', the apparent separate path-

ways for the transmission of the 2 types of pain, the difference in sensitivity of the different types of nerve fibre and the variation, in different individuals, of the topographical distribution of the nerve fibres in the nerve trunks (Sunderland, 1948), must all be taken into account. The situation may be further complicated by the factors suggested in Sinclair's (1955) 'Doctrine of Specific Energy', Dastur's (1955) submission that uncapsulated nerve fibres are not specific for any of the modalities of common sensation and that 'peripheral sensory innervation carries a large safety factor'. This suggests, by implication, that alternative pathways are available. Findings supported by the opinions of Gooddy (1957) in his submission that 'no single impulse or collection of impulses in a particular channel *is* a pain (touch, visual) impulse. It is a combination of impulses, in any number of anatomical (spatial) channels (fibres), of a particular rate of passage, and frequency, which provides the basic information from which we believe that perception is derived'; or, it must be added, in the case of complex syndromes, that symptoms are produced. These latter theories would suggest that there are no specific pathways for pain, or maybe for any other of the sensory or autonomic impulses (except perhaps the special senses of seeing, hearing and smelling).

Under normal circumstances, afferent impulses generated at the periphery, or by interference with a nerve fibre anywhere in its course, and having 'a specific dynamic pattern' are interpreted specifically by the sensorium and being noxious they will be interpreted as 'pain'. They follow customary* pathways and are accurately localized by the sensorium as originating in a particular 'arc of reference' unit.

Efferent impulses, generated by the same pathological cause, pass peripherally along associated fibres and are manifested as sensory changes in the dermatome, spasm or hyperalgesia in the myotome, scleratomal hyperalgesia, and vaso-motor or other autonomic symptoms at the points of their ultimate distribution.

In the neurocephalalgias with a thoracic component, it is noticeable that two types of pain are generally experienced. A sharp pain and a burning sensation, and where the autonomic system is involved, a throbbing ache. Concomitant autonomic manifestations are, *inter alia,* vertigo and disturbances of vision.

The route of conduction of the sharp pain is considered to be the A fibres and the burning or residual pain, the C fibres. Maher (1960) terms them the 'emergency pain' and the 'reminder pain' respectively. In three cases in the present series, stellate ganglion block relieved the 'emergency pain' and in Case 1 paraspinal block alleviated the 'reminder'. The combined procedures eliminated both types of pain. The opportunity to try these latter procedures in the migrainous type of case did not occur. (It is not easy to use patients as 'guinea-pigs' in general practice).

The observation that attacks of pain could only be produced in susceptible individuals when they were in a 'state of sensitivity' and that non-neurocephalalgic patients did not display any tender area in the T 7–9 region, nor did they respond to finger pressure or other stimulus applied in this area, by a complaint of pain in shoulder, neck or face, would suggest that disorganization of the intervertebral joint in this region, *in certain individuals,* can produce the 'state of sensitivity' and that the existence of this 'state of sensitivity' is a prerequisite for the development of paroxysms of neurocephalalgia of the hemi-cranial type. It is also suggested that some other factor superimposed on this 'state of sensitivity' will initiate an attack of neurocephalalgia specific for a particular individual at a particular time. This precipitating cause may be, *e.g.* a further disorganization, an allergic reaction, menstruation, or emotion.

The fact that only certain individuals are subjected to the onset of a 'state of sensitivity' produced by intervertebral joint disorganization points to the conclusion that these individuals are the possessors of a 'congenital' tendency comprising a peculiar interneural linkage which predisposes them to neurocephalalgic syndromes.

It is recognized that the neurocephalalgic type of headache is familial; almost without exception the cases in the series under discussion had such a history. It is, therefore, not beyond the bounds of possibility, that it is the 'congenital' tendency which is inherited and that the onset of attacks is subsequently triggered by some traumatic incident. Bickerstaff (1959) reports the occurrence of neurocephalalgia (autonomic facio-cephalalgia) in twins. The inheritable tendency might well be termed the neurocephalalgic diathesis.

* 'Customary' is here meant to refer to pathways selected by habit and experience which are normally accustomed to carrying impulses of certain characteristics.

CONCLUSIONS

To summarize, it would appear from the evidence provided by the cases cited that:

1. The present categorization of the neurocephalalgias and their being considered as different diseases is erroneous.

2. The apparent differences between the currently accepted categories lie only in the degree of intensity, the distribution and the complexity of the manifestations of a specific and similar lesion.

3. The manifestations of this specific lesion may vary in a particular individual, but only from mild to severe or simple to complex, rarely, if ever, in the opposite direction.

4. The causative lesion, at least in the cases cited here, is an intervertebral joint complex disorganization, a spondylo-dysarthria, in the T 7–9 region. As hyperaesthesia of the contra-lateral side to the hemicrania is rarely found, it is unlikely that zygapophyseal dysarthria is the cause of symptoms in these cases. It is more likely to be the costo-vertebral joint which is disorganised.

5. The initial effect of the dysarthria is the production of a 'state of sensitivity'. The acute exacerbations are superimposed on this condition.

6. Manipulative treatment directed at the correction of the dysarthria, when successfully performed, not only relieves an acute paroxysm, but also terminates the 'state of sensitivity'. In addition the period between bouts or attacks appears to be increased.

7. An inheritable neurocephalalgic diathesis occurs in certain families. This diathesis consists of the possession of an, as yet unidentified, specific neuronal linkage between one or other of the T 7–9 segments, the trigeminal nerve and, on occasions, the cervical spino-autonomic nerve complex.

8. The possession of a 'neurocephalalgic diathesis' is congenital, though its presence in the individual may only be made manifest following trauma.

It is fully appreciated that these submissions may be criticized on the grounds of the smallness of the series and the comparative shortness of the period of observation. It must be emphasized, however, that this paper does no more than report the observations made in the cases cited, and indicates, on anatomical and neurological grounds, that the theoretical basis put forward to justify the use of manipulative treatment in these cases is not unreasonable.

The identification of the thoracic component in neurocephalalgia, the observations that a paroxysm can be relieved *immediately* by a specifically directed manipulation, and that the intervening period between paroxysms can be lengthened in the individual case, are observed facts. The suggested nature of the mechanism involved is induced from these observed facts, and the known anatomical and neurological relationships between the structures in the vicinity of the intervertebral joint complex. It still remains subject to experimental proof or disproof, but it does appear to lie within the known laws of anatomy, neurology and pathology.

No claim is made that all neurocephalalgic syndromes have the suggested aetiology, but it is claimed that there is, at present, no acceptable alternative aetiological explanation of the phenomena observed in the 32 cases cited by the author.

It is not claimed that manipulative treatment will provide a permanent cure for what may be termed spondylodysarthric neurocephalalgias. The 'neurocephalalgic diathesis' consists of the possession, by the individual, of a particular anatomico-neural structure that provides the channels for the transmission of the impulses initiated by noxious stimuli in a particular intervertebral joint disorganization. This structure permits the production of referred symptoms, subjective and objective, in the terminal components of the 'arc of reference'. Obviously it cannot be removed by any manipulative procedure. These can only correct the precipitating dysarthria, as and when it occurs.

Bickerstaff (1959) suggests that a 'cure' which does not provide relief in 48 hours is not a cure. In the cases cited in this paper, more often than not, relief was obtained in a matter of minutes and the period of freedom from paroxysms extended from days to months.

Only extended investigation, over a more extensive field than that provided by one general practice, will show what proportion of neurocephalalgias are of spondylodysarthric origin and to what extent manipulation can be considered the treatment of choice.

SUMMARY

The suggestion is made that certain of the neurocephalalgias, including some cases of 'idiopathic' trigeminal neuralgia, atypical facial neuralgia, migraine and autonomic faciocephalalgia, are manifestations of a specific lesion in the intervertebral joint complexes of the T 7–9 segments.

Illustrative cases are cited.

The suggestion is made that a 'neurocephalalgic diathesis' exists, consisting of a congenital and inheritable specific anatomico-neural linkage between the T 7–9 spino-autonomic nerve complex in these segments, the trigeminal nerve and the cervical spino-sympathetic system.

Specifically directed spinal manipulation is the treatment of choice in these cases.

I am indebted to Dr. D. R. Stilwell, Jnr., for permission to reproduce 2 of his illustrations.

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PREPARATIONS AND APPLIANCES

DIANABOL

ANABOLIC SUBSTANCE

Dianabol is a new-type steroid which, when administered orally, exerts an intensive effect on protein metabolism. By promoting protein synthesis, **Dianabol** encourages the constructive metabolism of proteins. This leads to a positive nitrogen balance, to an increase in appetite and body-weight, and to an improvement in the patient's general condition. **Dianabol** also has a favourable effect on the calcium balance, causing increased deposition of calcium in the bones.

Indications:

Dianabol is indicated in all diseases and conditions in which it is desired to achieve an anabolic effect, i.e. to promote the constructive metabolism of proteins, and to strengthen the entire organism by exerting a general tonic effect.

Dianabol is accordingly suitable for use in the following cases:

Retarded convalescence following severe infections, operations, and confinements.

General physical deterioration or cachexia due to chronic infectious diseases, radiation therapy, or other causes.

States of debility and exhaustion.

Underweight and emaciated patients.

Symptoms of senescence associated with a decline in physical energy; pains in the back and lumbar region due to osteoporosis.

Osteoporosis, destructive bone diseases, delayed callus formation.

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Renal diseases (e.g. nephrosis, renal insufficiency).

Developmental and growth disorders in infants and children of all ages.

Administration and Dosage:

For adults the average initial dose is 10-20 mg. **Dianabol** daily. For maintenance therapy 5-10 mg. daily usually sufficient. The daily dosage is administered in 1-2 fractional doses. Depending on the state of the case.

Dosage for children:

Infants and children up to 2 years: 1 drop (=approx. 0.04 mg.) per kg. body-weight daily.

Children aged 2-5 years: 0.5-1 mg. daily.

Children aged 6-14 years: 1-2 mg. daily.

The **Dianabol** drops may be given in milk, fruit juice, or tea.

Administered in the recommended dosage, **Dianabol** produces as a rule no side effects. Children and adolescents should not be treated with **Dianabol** for periods of more than 4 weeks at a time, after which the treatment should be interrupted for 4-6 weeks.

Each tablet contains either 5 mg. or 1 mg. 17*a*-methyl-17*β*-hydroxyandrosta-1,4-diene-3-one. 1 c.c. of the drops contains 1 mg. **Dianabol.**

Packages:

Tablets of 5 mg.: Bottles of 20 and 100.

For paediatric use:

Tablets of 1 mg.: Bottles of 20.

Drops containing 1 mg. per c.c. (=30 drops): Bottles of 20 c.c.

CIBA (Pty.) Ltd. P.O. Box 5383, Johannesburg.

INTRAVAL

THIOPENTONE SODIUM SUPPOSITORIES

Maybaker (S.A.) (Pty.) Ltd., wish to announce the addition of a 750 mg. suppository to their range of **Intraval** brand thiopentone sodium suppositories. The complete range now consists of suppositories each containing 125 mg., 250 mg., 500 mg. or 750 mg. thiopentone sodium. The suppositories are indicated as a basal narcotic in pre-anaesthetic medication in children.

The suggested method of use is to administer the suppositories rectally 15 to 30 minutes before anaesthesia in a dosage of 20 mg. per kg./bodyweight.

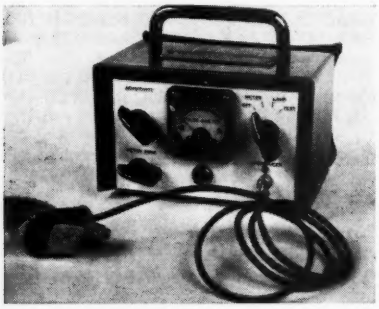
Further particulars may be obtained from Medical Department, Maybaker (S.A.) (Pty.) Ltd., P.O. Box 1130, Port Elizabeth.

PULSE-O-METER

Westdene Products (Pty.) Ltd. announce the introduction of a **Pulse-O-Meter** manufactured by E. J. Middleton (Electronics) Pty. Ltd. of Johannesburg.

The **Pulse-O-Meter** has been developed as an aid to the anaesthetist to give him a continuous indication of pulse rate during operations. Two types of transducers can be supplied, one for application to the thumb and the other for direct application to a convenient artery.

Indication of pulse rate is given on a meter and provision has been made for a lamp indication, so that the unit can be used for cardiac catheterization work when the patient is being X-rayed.

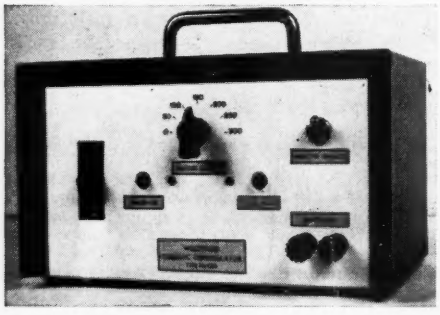


The complete unit is self-contained and operates from an internal battery. Battery test facilities have been incorporated so that the battery condition can be checked. It is housed in an attractive metal box with a suitable pouch for holding the transducer.

Other South African built instruments available from Westdene Products (Pty.) Ltd., manufactured by E. J. Middleton (Electronics) Pty. Ltd., are:

WESTPROD CARDIAC DEFIBRILLATOR

Operating Voltage: 220/250 v. A.C.
Output on Patient Circuit: 50, 100, 150, 200, 250, 300 v.
Impulse to Patient: 0.2 second's duration. Non-locking switch allows impulses to be repeated as required.



Safety automatic relay switch in patient's circuit for complete protection.
Electrodes: one spoon shaped, one flat round on insulated handles.
'*Power on*' *and* '*Impulse on*' indicators—neon lamps.
Size: 9″ x 6″ x 5½″. Weight ± 5 lb., fitted with carrying handle and mounted on rubber feet.
Housed in a red metal cabinet for easy identification.
S.A. Bureau of Standards Report for inspection on request.

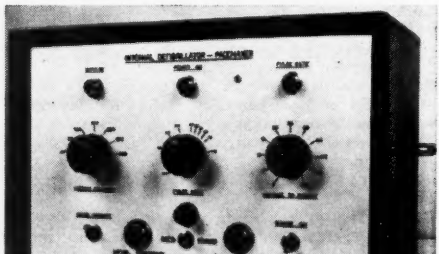
WESTPROD CARDIAC PACEMAKER

The Unit is a fully Transistorized Battery/Main's operated Pacemaker.
Battery operation comes into effect immediately in the event of a power failure, or when disconnected from main's power to transport patient from operating theatre to ward.
Built-in switch for checking battery voltage.
Rate of Stimuli ranges from 35 to 180 per minute and is selective.
Indicator bulb on the panel flashes with each stimulus.

Voltage output delivered to the heart is adjustable from 0 to 20 volts. The voltage selector is a calibrated dial.
Built-in dual-purpose meter is a novel feature used for:
(a) Checking the battery voltage.
(b) Indicating the actual current passed to the heart.
Its usefulness cannot be overestimated when considering the fact that after prolonged use the electrodes, or 'heart wires' attached to the heart become covered with scar tissue. This factor causes less current to pass to the heart. The meter indicates this decrease. The operator, by applying more output voltage, can then return the voltage to its original intensity.
Housed in a green metal cabinet, size 9″ x 6″ x 7″. The unit weighs only 5 lb. and is completely portable. Two insulated cardiac wires fitted with needles are supplied with each unit complete with electrode wires.

WESTPROD INTERNAL DEFIBRILLATOR–PACEMAKER

This is a *Combined Unit* consisting of a *Defibrillator* and a *Pacemaker*.
Both units operate off 220/250 v. 50 cycle A.C.
Defibrillator Output to Patient: 50-300 v.
Impulse Rate: 0.2 second's duration; non-locking switch allows impulses to be repeated as required.



Safety automatic relay switch in patient's circuit, affording complete protection.
Impulse on Indicator: Neon lamp.
Selector Switch: Defibrillator to Pacemaker.
Pacemaker Voltage to Patient: 0 to 50 v.
Pulse Rate to Patient: 35 to 130 per minute.
Pulse Rate Indicator: Neon lamp flasher.
Common Power Indicator: Neon lamp.
Both complete with electrodes and cardiac wire.